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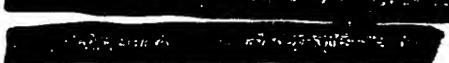
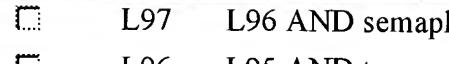
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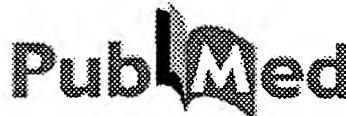
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Aneuploidy and cancer.

Sen S.

University of Texas, M.D. Anderson Cancer Center, Department of Laboratory Medicine, Houston 77030, USA. ssen@mdanderson.org

Numeric aberrations in chromosomes, referred to as aneuploidy, is commonly observed in human cancer. Whether aneuploidy is a cause or consequence of cancer has long been debated. Three lines of evidence now make a compelling case for aneuploidy being a discrete chromosome mutation event that contributes to malignant transformation and progression process. First, precise assay of chromosome aneuploidy in several primary tumors with *in situ* hybridization and comparative genomic hybridization techniques have revealed that specific chromosome aneusomies correlate with distinct tumor phenotypes. Second, aneuploid tumor cell lines and *in vitro* transformed rodent cells have been reported to display an elevated rate of chromosome instability, thereby indicating that aneuploidy is a dynamic chromosome mutation event associated with transformation of cells. Third, and most important, a number of mitotic genes regulating chromosome segregation have been found mutated in human cancer cells, implicating such mutations in induction of aneuploidy in tumors. Some of these gene mutations, possibly allowing unequal segregations of chromosomes, also cause tumorigenic transformation of cells *in vitro*. In this review, the recent publications investigating aneuploidy in human cancers, rate of chromosome instability in aneuploid tumor cells, and genes implicated in regulating chromosome segregation found mutated in cancer cells are discussed.

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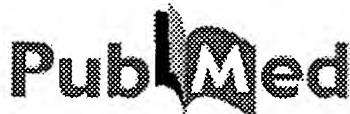
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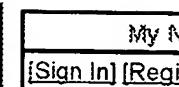
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Analysis of genomic and proteomic data using advanced literature mining.

Hu Y, Hines LM, Weng H, Zuo D, Rivera M, Richardson A, LaBaer J.

Institute of Proteomics, Harvard Medical School-BCMP, 240 Longwood Avenue, Boston, Massachusetts 02115, USA.

High-throughput technologies, such as proteomic screening and DNA micro-arrays, produce vast amounts of data requiring comprehensive analytical methods to decipher the biologically relevant results. One approach would be to manually search the biomedical literature; however, this would be an arduous task. We developed an automated literature-mining tool, termed MedGene, which comprehensively summarizes and estimates the relative strengths of all human gene-disease relationships in Medline. Using MedGene, we analyzed a novel micro-array expression dataset comparing breast cancer and normal breast tissue in the context of existing knowledge. We found no correlation between the strength of the literature association and the magnitude of the difference in expression level when considering changes as high as 5-fold; however, a significant correlation was observed ($r = 0.41$; $p = 0.05$) among genes showing an expression difference of 10-fold or more. Interestingly, this only held true for estrogen receptor (ER) positive tumors, not ER negative. MedGene identified a set of relatively understudied, yet highly expressed genes in ER negative tumors worthy of further examination.

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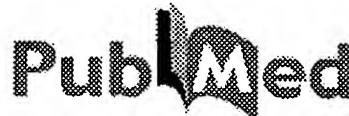
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Aneuploidy and cancer.

Sen S.

University of Texas, M.D. Anderson Cancer Center, Department of Laboratory Medicine, Houston 77030, USA. ssen@mdanderson.org

Numeric aberrations in chromosomes, referred to as aneuploidy, is commonly observed in human cancer. Whether aneuploidy is a cause or consequence of cancer has long been debated. Three lines of evidence now make a compelling case for aneuploidy being a discrete chromosome mutation event that contributes to malignant transformation and progression process. First, precise assay of chromosome aneuploidy in several primary tumors with *in situ* hybridization and comparative genomic hybridization techniques have revealed that specific chromosome aneusomies correlate with distinct tumor phenotypes. Second, aneuploid tumor cell lines and *in vitro* transformed rodent cells have been reported to display an elevated rate of chromosome instability, thereby indicating that aneuploidy is a dynamic chromosome mutation event associated with transformation of cells. Third, and most important, a number of mitotic genes regulating chromosome segregation have been found mutated in human cancer cells, implicating such mutations in induction of aneuploidy in tumors. Some of these gene mutations, possibly allowing unequal segregations of chromosomes, also cause tumorigenic transformation of cells *in vitro*. In this review, the recent publications investigating aneuploidy in human cancers, rate of chromosome instability in aneuploid tumor cells, and genes implicated in regulating chromosome segregation found mutated in cancer cells are discussed.

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Proteome analysis: biological assay or data archive?

Haynes PA, Gygi SP, Figgeys D, Aebersold R.

Department of Molecular Biotechnology, University of Washington, Seattle 98195, USA.

In this review we examine the current state of proteome analysis. There are three main issues discussed: why it is necessary to study proteomes; how proteomes can be analyzed with current technology; and how proteome analysis can be used to enhance biological research. We conclude that proteome analysis is an essential tool in the understanding of regulated biological systems. Current technology, while still mostly limited to the more abundant proteins, enables the use of proteome analysis both to establish databases of proteins present, and to perform biological assays involving measurement of multiple variables. We believe that the utility of proteome analysis in future biological research will continue to be enhanced by further improvements in analytical technology.

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